REPORT OF DR. RAYMOND D. HARBISON

1. I am a Board Certified Toxicologist, pharmacologist, human health risk assessor, and Professor in the College of Public Health at the University of South Florida, Professor of Pharmacology and Pathology in the College of Medicine, and Professor of Pharmaceutical Sciences in the College of Pharmacy at the University of South Florida, and adjunct Professor of Medicine at the College of Medicine, State University of New York at Buffalo. I earned a doctorate in Pharmacology/Toxicology at the University of Iowa, College of Medicine in 1969.

2. I served on the faculty of Tulane Medical School, New Orleans, Louisiana from 1969 to 1971 and Vanderbilt Medical Center, Nashville, Tennessee from 1971 to 1981. I served as Director of the Interdisciplinary Toxicology Program at the University of Arkansas for Medical Sciences, Little Rock, Arkansas from 1981 to 1988. I was a Professor of Pathology, Toxicology, and Pharmacology at the Health Science Center at the University of Florida, Gainesville, Florida from 1988 to 1996. I have served on the editorial boards of scientific journals, including *Fundamental and Applied Toxicology*, *Environmental Health Sciences*, *Teratogenicity, Mutagenicity, & Carcinogenicity, Journal of Emergencies, Trauma, and Shock, International Journal of Critical Illness and Injury Science, Open Toxicology Journal,* and *Research Communications in Pharmacology and Toxicology*. I have served on advisory committees for the National Institute of Mental Health, Drug Abuse Warning Network (DAWN), National Academy of Sciences, National Research Council, American Cancer Society, the United States Environmental Protection Agency, National Institute of Occupational Safety and Health, National Institute of Environmental Health Sciences, and National Institute for Drug Abuse.

3. I have authored over 190 scientific articles. My membership in scientific societies includes the Society of Toxicology, Society for Risk Analysis, Teratology Society, American Society for Pharmacology and Experimental Therapeutics, and the New York Academy of Sciences. My experience includes research and teaching related to general pharmacology and toxicology, evaluating potential mechanisms of toxicity in association with alleged health effects, and evaluating patients potentially affected by drugs and chemicals who present to the Occupational and Environmental Medicine Consultation at the University of South Florida. The attached curriculum vita details my professional credentials.

4. I have been retained by counsel for Waste Management, Inc., Waste Management of Texas, Inc., and McGinnes Industrial Maintenance Corporation. I have reviewed the following materials that include but are not limited to Plaintiffs' (Harpster) Original Petition, Plaintiffs' (Pho) Second Amended Petition, Plaintiffs' (Harpster) Fourth Amended Original Petition, Plaintiffs' (Pho) Third Amended Petition, Plaintiff Harris County's Second Amended Petition, medical records of Tommy Nguyen, Thuy Mai, Alexander Pham, Trang Au, James Tran, Johnny Tran, Jimmy Tran, Jackson Tran, and Khanh Tran, Public Health Assessment report (2012), Plaintiffs' Interrogatory Responses including James Tran, Trang Au, Jimmy Tran, Alexander Pham, Thuy Mai, Joanne

Nguyen, Johnny Tran, and Hong Van Pham, and scientific and medical literature. I am aware a claim is made that various health conditions of certain plaintiffs were caused as a result of exposure to various constituents, including dioxins, allegedly released from waste sites near the San Jacinto River. In addition, a claim is made that medical monitoring is required for certain plaintiffs as a result of alleged injuries that currently exist.

5. It is my conclusion after reviewing the various records and reports that the methodologies and procedures used to reach the conclusion that constituents released from the former disposal sites caused the conditions of plaintiffs are not based on valid and reliable scientific principles. In addition, the information relied upon and advanced as supportive of the claims is not generally accepted by the scientific community as supplying an adequate basis for the conclusions reached. Moreover, the scientific community does not generally accept these methods. The mere presence of various constituents at levels above government guidelines is not sufficient evidence that these constituents caused various conditions in any specific individual. Further, there are dozens of dioxins and other constituents. The specific putative agents claimed to be the cause of the specific conditions of specific plaintiffs and require medical monitoring have not been identified.

6. The claim is made that alleged disposal site constituents caused specific health conditions of certain plaintiffs and increased their health risks. To arrive at conclusions relevant to this claim, there must first be scientifically relevant, reliable, and applicable evidence of general causation, that is, whether the level, frequency, and duration of alleged dioxins and constituents exposures claimed to be encountered by plaintiffs was capable of causing these specific conditions. Subsequently, there must be consideration of specific causation, that is, whether the exposure alleged to be encountered by each plaintiff caused sub-cellular or other physiological changes that actually caused their specific conditions. In order to reach a valid conclusion, opinions must be based upon reliable evidence, including the following:

a. There was a harmful amount of specific waste site constituents in the environment of each plaintiff;
b. that each of them was exposed to a harmful amount of specific waste site constituents;
c. that the frequency and regularity of exposure and dose of specific constituents was sufficient to cause their specific conditions and risks; and
d. that the alleged specific waste site constituents exposures encountered by plaintiffs actually caused their specific conditions.

No reliable methodology has been used to determine whether alleged waste site constituent exposures caused subcellular or other physiological changes in plaintiffs that caused increased health risks. No objective evidence of any waste site constituent-induced sub-cellular or other physiologic changes has been provided for plaintiffs. The risk claims are speculative because they have not been quantified or validated. The level, frequency, and duration of alleged waste site constituent exposure for each plaintiff has not been quantified. No specific exposure evaluation(s) has been provided for each plaintiff. The claim is alleged exposure to waste site constituents caused various health conditions and increased risk of developing other health conditions. However, the increased risk has not been quantified for each specific plaintiff and compared with

other risks and their inherent risks of cancer and/or other conditions. No comparative risk analysis is presented for plaintiffs.

No dose-response relationship has been described. Absent opinions about dose-response relationships, it is not possible to determine whether any plaintiffs encountered sufficient exposure to increase their individual risk of developing adverse health conditions. Further, not all plaintiffs would encounter the same waste site constituent exposures, therefore each plaintiff would need to be evaluated separately to determine if the exposure increased their individual risk of developing adverse health conditions. All plaintiffs' exposures are not likely to be equal or the same, therefore considering all plaintiffs the same for assessing their individual risk is not justified.

The claim relies on sampling data that exceeds government screening levels. Regulatory levels are not designed to predict adverse health effects; rather they are designed to maintain environmental levels far below the threshold of toxicity to prevent the risk of illness in exposed persons. Often times, a regulatory level is as much as 1,000 times lower than an exposure level which has been shown to cause no adverse effects in test animals or epidemiological studies. If an exposure exceeds a regulatory screening level, it does not indicate that exposed persons are at any risk of developing disease, as the exposure may remain orders of magnitude below an exposure level that has been demonstrated to cause no harm. The claim is based on the implication that exceeding a regulatory screening limit indicates that a population is consequently at risk of harm from that exposure – an opinion that is wholly incorrect and not supported by toxicological and epidemiological evidence.

The underlying predicate of any determination of the cause of a human ailment or disease and/or health risk is that medical science understands the dose response relationship and physiological process by which a particular disease or syndrome develops and what factors cause the process to occur. Based on such predicate knowledge, it may then be possible to determine the cause and/or risk associated with various exposures. The claim has not offered a reliable or credible explanation for how the alleged exposure to waste site constituents caused the conditions and/or increased the health risk of each individual plaintiff. In the absence of a quantified exposure assessment for each plaintiff, any opinions about the cause of their individual health conditions and/or health risks alleged to be associated with waste site constituent exposures are not reliable.

7. The claim describes general associations but has not provided a relevant, reliable, and applicable evaluation of the exposure and conditions of each plaintiff. No relevant, reliable, and applicable exposure assessment has been provided for each plaintiff. The specific conditions alleged to be caused by specific waste site constituents have not been identified. Instead, the alleged conditions have been consolidated with a list of alleged constituent exposures. The allegation is that these waste site constituents caused a variety of conditions in plaintiffs. This is speculation. Finding samples with waste constituents above a government guideline does not establish a cause and effect relationship for any specific condition of any specific plaintiff. The level, frequency, and duration of specific alleged waste site constituent exposure required to cause the specific conditions of each plaintiff has not been provided. The frequency and regularity of plaintiffs' encounter with specific waste site constituents has not been described. No dose response claims for any specific constituent and whether plaintiffs' exposures exceeded a

threshold necessary to cause a specific condition or increase the risk of developing a specific condition have been provided. Simply claiming the presence of various waste site constituents is not sufficient evidence that the dose resulting from the alleged exposure can cause any specific condition. Exposure is only the opportunity for contact. Dose is what enters the body. Exposure to many of the alleged waste site constituents is common for individuals not living in the areas near the waste sites. The allegation that any dose of alleged constituent can cause any of the alleged conditions and increase risk is speculation. There are many individual factors that can affect their individual risks. In sum, no generally accepted methodology was used to arrive at the claim that any specific alleged waste site constituent caused any specific condition in any specific plaintiff and this claim therefore, is not scientifically reliable.

8. Further, from my training and experience, I am thoroughly familiar with and have reviewed the scientific and medical literature concerning dioxins and other alleged waste site constituents and their potential human health effects and risks. In addition, from my training and experience, I am aware of available scientific literature describing the accepted scientific methodology required to determine the cause and risk of a human disease or ailment. I am also aware of the requirement to apply this same methodology to establish any causal link between a claimed exposure and an alleged disease and/or risk. This methodology must be followed in order to differentiate a disease or risk associated with alleged waste site constituents exposures versus other causes and risks. I am also aware of the requirement to apply this same methodology to establish any causal link between alleged waste constituents exposures and alleged diseases, in order to differentiate an ailment caused by alleged waste site constituents from an ailment that occurred spontaneously, by chance, or was caused by some other substance or exposure. Not following the scientific methodology may result in arbitrary and incorrect associations because of individual bias, confounders, and/or failure to use a precise and objective comparison.

This generally accepted methodology was not used to support this claim. No comparative risk analysis was used to determine other risks for the conditions and complaints of plaintiffs. No relevant, reliable, and applicable epidemiologic literature has been provided to support the claim that alleged waste site constituents caused the health conditions of plaintiffs and increased their risk of future health conditions.

9. In any situation involving exposure to a substance, where the alleged exposure is followed by an allegedly observable physiological change or deficit, the ultimate problem confronting the practitioner attempting to attribute a cause lies in determining whether there is a scientific basis for concluding that the observed effect would not have occurred in the absence of the exposure. Various individuals and organizations have attempted to devise a methodology sufficient for linking changes occurring in a specific individual following specific exposures. There is general agreement that in the absence of certain information, no valid conclusion as to cause and effect is possible in a specific individual's case.

The principles and methods of toxicology must be applied when attempting to determine the potential toxic effects of alleged waste site constituents. The scientific community requires the following minimal information before expressing any reasonable medical or scientific probability relating an alleged waste site constituent exposure and an observed effect in a specific individual:

a. Exposure to a putative substance must be documented.

b. The exposure must occur in such a fashion that the substance is temporally eligible to be the cause of the observed effect.

c. The exposure level must be documented at a level capable of inducing a known effect.

d. The observed effect, whether acting directly on the target organ, or indirectly through alteration of body chemistry or function, must be satisfactorily linked to the observed effect in the target organ.

e. An effect suspected of being responsible, either directly or indirectly for injury to a specific organ must have been replicated in a general population upon identical exposure.

f. Confounding variables, such as concomitant exposures, life style, intrinsic factors, or effects caused by infectious diseases, must be eliminated as potential causal or contributing factors.

g. If the latency period (the time between exposure and alleged effect) is extended, some plausible explanation for delay of onset of the disease process must be present, through data from either similarly exposed populations or other sources.

h. The specific effect from the putative constituent must be demonstrated as occurring in the specific individual involved. In cases where no effect can be demonstrated other than injury to a target organ, no conclusion can be drawn unless specific cytotoxicity affecting the target organ can be demonstrated.

i. A consistent pattern of identical effects under controlled circumstances must be demonstrated (literature precedence).

J. A consistent morphologic pattern under controlled circumstances (or a pathognomonic effect) must be demonstrated and existence of the specific morphologic pattern confirmed in the individual case under consideration.

k. Epidemiologic and bioassay tests must be supportive.

The minimum information to be able to determine that exposure from the alleged waste site constituents caused sub-cellular or other physiological changes in plaintiffs that caused their individual health conditions and alleged future risks of health conditions has not been provided. Each plaintiff's level, frequency, and duration of alleged waste site constituent exposure must be evaluated to determine whether a cause and effect relationship exists between exposure and each of their specific health conditions and their individual alleged increased risk of specific health conditions. At present no specific individual exposure analysis or risk assessment has been provided for any plaintiff.

The background occurrence of alleged conditions have not been quantified. No comparative risk analysis has been provided to compare the background risk of developing these conditions with the possible risk of developing the condition as a result of alleged exposure to waste site constituents. Absent this comparative risk analysis, the background risk confounder cannot be eliminated. In addition, incomplete analysis of the specific conditions of specific plaintiffs introduces significant uncertainty in any conclusions made from the available data.

10. Any practitioner claiming to know the cause of an individual's condition has an obligation to describe the methodology used to arrive at that conclusion. The level, frequency, and duration of specific waste site constituents required to cause each specific health condition has not been defined. Further, the features of these conditions that are necessary to have been caused by waste site constituent exposures have not been described. The frequency and regularity of waste site constituent exposure that is necessary to cause the individual specific conditions of plaintiffs' have not been quantified. The temporal relationship that is necessary to conclude that alleged waste site constituents exposures caused each of these specific health conditions has not been described. Relevant, reliable, and applicable epidemiologic literature has not been provided to support a consistent pattern of similar effects as seen in these plaintiffs under similar exposure conditions as claimed for these plaintiffs.

11. Finally, the scientific basis provided to support the claim that alleged waste site constituents exposures caused plaintiffs conditions is wholly inadequate. Even under the minimal standards described herein, there is insufficient scientific basis for concluding that alleged waste site constituents exposures, as alleged for plaintiffs, caused their health conditions and increased their risk of future adverse health conditions.

12. I reserve the right to alter my opinions and conclusions should additional information become available. All of my opinions are held to a reasonable degree of toxicological, scientific, and medical certainty.

Raymond D. Harbison, MS, Ph.D.
August 15, 2013

References

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CURRICULUM VITAE

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Date and Place of Birth: January 1, 1943, Peru, Illinois

EDUCATION

Drake University, Des Moines, Iowa - 1961 to 1965, B.S.

University of Iowa, Iowa City, Iowa - 1965 to 1967, M.S. (Pharmacology) -
Thesis: Induced hyperbilirubinemia and a quantitative method or analysis of diazotized bilirubin.

University of Iowa, College of Medicine, Iowa City, Iowa - 1967 to 1969, Ph.D. (Pharmacology/Toxicology) - Dissertation: Studies on the mechanism of teratogenic action and neonatal pharmacology of diphenylhydantoin.

SUMMARY OF EXPERIENCE

2012
Human Health Peer Review Committee
Florida Department of Environmental Protection

2011- Present
Professor of Pharmceutical Sciences
College of Pharmacy
University of South Florida, Tampa

2011-Present
Editorial Board, International Journal of Critical Illness and Injury Science

2009-Present
Editorial Board, Journal of Emergencies, Trauma, and Shock

2007-Present
Editorial Board, The Open Toxicology Journal

2004-Present
Department of Health
State of Florida
Environmental Public Health Tracking Advisory Committee

2004-Present

College of Medicine
University of South Florida
Occupational Medicine Residency Program
Toxicology Rotation

2003-Present
Associate Member
H. Lee Moffitt Cancer Center & Research Institute
Tampa, FL

2003-2005
Chairman, Public Health Oversight Committee
Saudi Arabia

2002-2009
RESEARCH PROFESSOR, Center for Environmental Diagnostics and Bioremediation
The University of West Florida
Pensacola, FL

2001-2005
Training Director, NIOSH ,Hazardous Substances Continuing Education Training
NIOSH - Education and Research Center, University of South Florida

2000-2010
Brownfield Advisory Board- Hillsborough County, Florida

2000-Present
Editorial Board- Research Communications in Pharmacology and Toxicology

1999-2005
Vice Chair, Technical Advisory Committee, Brownfield Advisory Board, City of Clearwater, Florida

1998-Present
Toxicologist, Research Service, Haley Veterans Administration Hospital, Tampa, Florida

1998-Present
Professor, Department of Pathology, College of Medicine, University of South Florida

1995-Present
Adjunct Professor, Department of Medicine, College of Medicine, State University of New York at Buffalo

1995-Present
Professor, Department of Pharmacology and Therapeutics, College of Medicine, University of South Florida

1995-Present
Professor, Department of Environmental and Occupational Health, College of Public Health, University of South Florida

1995-Present
Director, Environmental/Occupational Risk Analysis & Management Research, College of Public Health, University of South Florida

1996-2005
Director, Certification of Hazardous Materials Handlers Review Course and Examination

1994-Present
National Institute of Health, Reviewers Reserve

1994-1995
Advisory Board of Center for Training, Research, and Education for Environmental Occupations (TREEO), University of Florida

1993-1997
American Cancer Society Study Review Group (Florida)

1993-1997
Academy of Toxicological Sciences, Board of Directors

1992-1997
Advisory Board of Earth 2020, the University of Virginia's Center for Environmental Policy

1991-1995
Faculty, Superfund University Training Institutes, East Tennessee State University and University of Virginia

1991-1995
Science Advisory Board Consultant, Environmental Health Committee, United States Environmental Protection Agency

1990-1995
Professor, Department of Pathology, College of Medicine, University of Florida

1989-1995
Director, Laboratory for Environmental and Human Toxicology, University of Florida

1989-1997
Speciality Staff, St. Vincent Infirmary Medical Center, Little Rock, Arkansas

1989-1996
Board of Advisors, The Environmental Institute

1989 - 1994

National Institute of Drug Abuse, Pharmacology II Study Review Group

1988-1995
Professor, Department of Pharmacology and Therapeutics, College of Medicine, University of Florida

1988-1995
Professor, Department of Physiological Sciences, Health Science Center, University of Florida

1987-1997
Clinical Professor, Department of Preventive Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin

1986-1990
National Institute of Environmental Health Sciences Study Review Group

1984-1985
Chairman, National Institute of Occupational Safety and Health Study Review Group

1982-1986
Editorial Board, Fundamental and Applied Toxicology

1980-1990
Society of Toxicology Liaison with Teratology Society

1980-1988
Professor, Department of Pharmacology, University of Arkansas for Medical Sciences

1980-1984
National Institute of Occupational Safety and Health Study Review Committee

1979-Present
Editorial Board, Teratogenicity, Mutagenicity, Carcinogenicity

1977-1980
Director, Toxic Substance Control Laboratory and Associate Professor, Department of Pharmacology and Biochemistry, School of Medicine, Vanderbilt University, Nashville, Tennessee

1977-1980
Professional Affairs Committee - American Society for Pharmacology and Experimental Therapeutics

1977-1980
National Institute on Drug Abuse - Review of DAWN (Drug Abuse Warning Network)

1977-1979
Graduate Education Committee, Vanderbilt University

1977-1978

National Research Council Committee to Review Scientific Program of National Center for Toxicological Research

1976-1980
Editorial Board, Environmental Health Sciences

1976-1977
National Academy of Science, Advisory Center for Toxicology - Revision of Toxicity Testing Procedures for Consumer Protection Agency

1975-1976
Technical Committee of the Society of Toxicology, Chairman

1975-1976
National Institute on Drug Abuse Center Review Committee

1974-1980
Editorial Board, International Journal of Addictive Diseases

1974-1979
Vanderbilt Medical Center, Animal Care Committee

1974-1978
Consultant, U.S. Congressional Committee on Safety Assessment of Chemical Additives & Drugs

1974-1975
Standing Policy Committee on Biomedical Sciences, Vanderbilt School of Medicine; National Institute on Drug Abuse, Clinical Behavioral Review Committee

1974-1975
Co-Chairman, Technical Committee of the Society of Toxicology

1972-1976
Assistant Professor of Pharmacology and Biochemistry, Vanderbilt University School of Medicine

1971-1975
National Institutes of Mental Health-Narcotic Addiction and Drug Abuse Review Committee, Biomedical-Pharmacology-Toxicology

1971-1972
Assistant Professor, Department of Pharmacology, Tulane Medical School

1971-1972
Director of Teratology Section, Laboratory of Environmental Health, Department of Medicine, School of Medicine, Tulane University

1969-1970
Instructor of Pharmacology, Tulane Medical School

1965-1969
USPHS Trainee, University of Iowa, Department of Pharmacology, College of Medicine, Iowa City, Iowa

PROFESSIONAL SOCIETIES

Rho Chi Honorary Pharmacy Society
Sigma Xi (Promotion of Research in Science)
American Association for the Advancement of Science
Teratology Society
Society of Toxicology
American Society for Pharmacology and Experimental Therapeutics
New York Academy of Science
Society for Risk Analysis
American Industrial Hygiene Association

AWARDS

1978
Society of Toxicology Achievement Award

CERTIFICATION

1982
1987
1992
1997
2002
2006
Certified in General Toxicology
Registration
Registered Professional Industrial Hygienest
1999

INDUSTRIAL EXPERIENCE

- Occidental Oil - Worker safety in oil shale production
- Shell Development Corporation - Pesticide use and safety
- Petrolite Corporation - Health assessment of waste incineration methods
- Monsanto Corporation - Chemical mutagenesis and the workplace, environmental assessment of PCB pollution
- American Academy of Industrial Medicine - Women in the workplace
- Tennessee Occupational Safety Health Administration - Industrial toxicology training course

- Society Organic Chemical Manufacturing Association - Chemical carcinogenesis
- Sanitary Corporation of America - Worker safety at industrial residue landfill sites
- Ethyl Corporation - Chemical-induced mutagenesis and teratogenesis in the workplace
- State of Kentucky Bureau of Natural Resources - Toxicology training course for solid waste management personnel
- U.S. Environmental Protection Agency - Chemistry and toxicology of hazardous materials training course for spill management personnel
- Ecology and Environment, Inc. - Health and safety program advisor
- Texaco - Toxicology consultant
- Hooker Chemical Company - Evaluation of health effects at Love Canal
- Chemical Manufacturers Association - Technical review of health effects of PCBs
- American Petroleum Institute - Comments to EPA concerning Resource Conservation Recovery Act
- IBM Corporation - Reproductive hazard assessment
- U.S. Environmental Protection Agency - Rebuttable presumption review of pesticides (FIFRA)
- United States Department of Agriculture - Review of aerial pesticide applications
- State of Georgia Department of Environmental Protection - Toxicology training course for emergency environmental incident management
- State of North Carolina Environmental Resource Management Division - Toxicology training course for environmental management and public health personnel
- Exxon - Review of teratogenic hazard of benzene exposure
- U.S. Department of Justice - Evaluation of environmental and public health problems associated with Price Landfill, Atlantic City, New Jersey
- U.S. Department of Justice - Evaluation of environmental and public health problems associated with Bridgeport Oil and Rental contamination of the Delaware River, Bridgeport, New Jersey
- U.S. Environmental Protection Agency - Evaluation of the environmental impact of dredging of the Hudson River for PCBs, New York

- U.S. Environmental Protection Agency - Evaluation of environmental and public health problems associated with LiPari Landfill in New Jersey
- Velsicol Chemical Corporation - Evaluation of health problems associated with Hardeman County Landfill
- U.S. Environmental Protection Agency - Review of environmental and public health information associated with Denny Farm Site, Verona, Missouri
- U.S. Environmental Protection Agency - Review of environmental and public health information associated with Rose Park Landfill, Salt Lake City, Utah
- U.S. Environmental Protection Agency - Evaluation of environmental and public health problems associated with Taylor Road Landfill, Tampa, Florida
- Dow Chemical Company - Evaluation of health problems associated with a degreasing operation in Tyler, Texas
- United States Environmental Protection Agency - Training course director for Toxicology and Risk Assessment for eight southeastern states. (Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, Tennessee)
- Monsanto Chemical Company - Evaluation of adverse health effects associated with PCB contamination of feed grain in Michigan
- American Bar Association - Short course concerning the role of expert testimony in environmental litigations.
- Gates Energy - Evaluation of the toxicity of metal hydrides
- Diversitech/Gen Corp. - Evaluation of mortality among PCB exposed tire and plastic fabricators
- BIC - Evaluation of reproductive workplace hazards
- Breed Industries - Assessment of health risks associated with airbags

GRANT SUPPORT

Effect of Environmental Toxicants on Perinatal Development	ES00782	1970-1982
Effect of Marijuana on Perinatal Development	DA00141	1971-1974
Schleider Foundation Developmental Toxicology		1970-1972
Clinical Pharmacology - Toxicology Center	GM15431	1975-1981
Environmental Toxicology Center	ES00267	1972-1981

Synthesis and Study of New Chelating Agents	ES01018	1975-1981
Life Insurance Research Fund		1970-1981
National Conference on Control of Hazardous Materials Spills	EPA	1977-1978
Wampole Laboratories - Development of a Prenatal Diagnostic Aid for Neurotube Defects		1979-1981
Introduction to Hazardous Materials Incidence Responses and Environmental Hazards Evaluation	EPA	1980-1981
Environmental Toxicant Effects on Perinatal Development	ES02824	1981-1986
Study of the Reproductive Toxicity of Selected Chemicals	NF	1985-1987
Studies of Chemical-Induced Toxicity and Stress	ES05216	1988-1994
Studies of Isomer Specific PCB-Induced Toxicity	*Cooperative Agreement	1989-1991
Mechanism of Cocaine-Induced Liver Toxicity	NIDA	1990-1991
Methamphetamine-Induced Toxicity	NIDA	1991-1994
Toxicology Support	**DER/DEP	1991-1995
Radon Risk Assessment	HUD	1994-1996
Pediatric Formula Evaluation	Mead-Johnson	1994-1996
Halocarbon Toxicity	COPH	1995-1996
Assessment of Risk Associated with Vitrified Materials	DOE	1996-1997
Evaluation of Airbag Safety	Breed Industries	1997-1998
Adrenergic Modulation of Stress-Induced changes in	NIOSH-Center for	1999-2000

Pesticide Toxicity	Agricultural Research And Education	
Pesticide-Induced Toxicity	NIOSH	1999-2000
PAH-induced Cellular Changes	NIOSH	2000-2001
Environmental Health Monitoring	General Hlth Systems	2000-2003
Florida Biomonitoring	CDC	2001-2003
Arsenic Physician's Workgroup	FL DOH	2001-2002
Adrenergic Alterations of Toxins-Toxicants	DOD	20001-2003
Clinical Toxicology Evaluation of Health Among Residents of Escambia County, FL	CDC	2002-2004
Assessment of Public Health Risks of Blue/Green Algae Cyanobacter	FL DOH	2002-2003
Evaluation of Toxins on DNA	DOD	2003-2004
Brain's DNA Repair Response to Neurotoxicants	DOD	2004-2006
Florida Bioterrorism & Disaster Preparedness Consortium	HRSA	2005-2008
USF & University of Florida Colleges of Pharmacy & Veterinary Medicine Community Medical Trust Fund		2006-2009
Pesticide Biomonitoring	NIOSH	2009-2012
Evaluation of mechanisms of toxin-induced toxicity	DOD	2009-2010

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ES - National Institute of Environmental Health Sciences COPH-College of Public Hlth
DA - National Institute on Drug Abuse DOE- Dept of Energy
GM - National Institute of General Medical Sciences DOD- Dept of Defense
EPA - United States Environmental Protection Agency DOH- Dept of Health
NF - National Foundation March of Dimes
NIDA - National Institute on Drug Abuse HUD - Housing and Urban Development
NIOSH-National Institute of Occupational Safety & Health CDC-Ctr Disease Control
HRSA- Health Research Services Administration

TEACHING EXPERIENCE

Medical Toxicology	Vanderbilt Medical Center Second Year Medical Pharmacology
Developmental Pharmacology	Vanderbilt Medical Center
Drug Metabolism	Two Hour Graduate Level Course Vanderbilt Medical Center
Toxicology	Two Hour Graduate Level Course

	Vanderbilt Medical Center
Medical Toxicology	Second Year Medical Pharmacology University of Arkansas for Medical Sciences
Advanced Toxicology	Two Hour Graduate Level Course University of Arkansas for Medical Sciences
Oncology	Two Hour Graduate Level Course University of Arkansas for Medical Sciences
Chemical Carcinogenesis	Second Year Medical Pathology University of Florida, College of Medicine
Medical Toxicology	Second Year Medical Pharmacology University of Florida, College of Medicine University of South Florida, College of Medicine
Mechanism of Chemical-Induced Toxicity	Two Hour Graduate Level Course University of Florida, College of Medicine
Introductory Toxicology	Three Hour Graduate Level Course University of Florida, College of Medicine
Health Implications / Aspects of Chemical Issues	Superfund University Training Institute East Tennessee State University, University of Virginia, Environmental Protection Agency
Risk Assessment • Industrial • Occupational •Environmental	Three Hour Graduate Level Course University of South Florida, College of Public Health
Occupational and Environmental Medicine	Two Hour Graduate Level Course University of South Florida, College of Public Health
Biomonitoring	Two Hour Graduate Level Course University of South Florida, College of Public Health
Protecting Public Health: Bioterrorism & Biodefense	Three Hour Graduate Level Course University of South Florida, College of Public Health
Occupational Risk Assessment	Three Hour Graduate Level Course University of South Florida, College Of Public Health
Environmental Risk Assessment	Three Hour Graduate Level Course University of South Florida, College

	Of Public Health
Hazardous Materials and Hazard Communication	Three Hour Graduate Level Course University of South Florida, College Of Public Health

CONTINUING EDUCATION:

National Hazardous Materials Training Course	Eight Hours (Toxicology)
Hazardous Waste Management	Four Hours (Toxicology)
Toxic Substance Control	Eight Hours (Toxicology)
Environmental Protection Agency, Region IV, Health and Safety Training School	Six Hours (Toxicology)
Industrial Toxicology	Five Hours (Carcinogenesis, Teratogenesis)
Forensic Medicine	Three Hours (Toxicology)
Toxicology and Risk Assessment	Eight Hours
Certification for Hazardous Materials Managers	Twenty Four Hours (NIOSH - ERC)
Neurology and Solvent Encephalopathy	Eight Hours (NIOSH - ERC)
Toxicology and Risk Assessment	Eleven Hours (NIOSH - ERC)

GRADUATE TRAINING - PREDOCTORAL

	Year Degree Conferred	Present Address
Bernardo Mantilla-Plata, Ph.D.	1972	University of Antioqua Department of Toxicology Medellin, Columbia
Michael Stevens, Ph.D.	1973	Monsanto Chemical Company Toxicology Department St. Louis, Missouri
Richard W. Freeman, Ph.D.	1980	Ecology and Environment, Inc. Tallahassee, Florida
Michael E. Fant, M.D., Ph.D	1980	Department of Pediatrics University of Texas Houston, Texas

Adeline Smith, Ph.D.	1982	National Institute of Health Div. of Molecular Toxicology Bethesda, Maryland
James Jernigan, Ph.D.	1983	Amoco Chicago, Illinois
Christopher Teaf, Ph.D.	1985	Florida State University Center for Biomedical and Hazardous Waste Research Tallahassee, Florida
Felix Adatsi, Ph.D.	1986	Department of Natural Resources State of Michigan Lansing, Michigan
M. Ann Clevenger, Ph.D.	1987	EPA Washington, D.C.
Glenn C. Millner, Ph.D.	1987	Environmental Consultant Little Rock, Arkansas
Henry F. Simmons, M.D., Ph.D.	1988	Division of Clinical Toxicology University of Arkansas for Medical Little Rock, Arkansas
Mary Alice Smith, Ph.D.	1989	University of Georgia Athens, Georgia
Hudson K. Bates, Ph.D.	1989	Research Triangle Institute Research Triangle Park, NC
Roland Garipay, MSPH	1998	United States Navy Norfolk, Virginia
Todd Stedeford,Ph.D.	2000	U.S.E.P.A. Washington, DC
Deborah Price, Ph.D.	2001	Environmental Health Hillsborough County, FL
Rony Francois, M.D., Ph.D.	2003	Secretary of Health State of Florida
Kelly Hall, Ph.D.	2005	Baush & Laumb Tampa, Florida
G. Scott Dotson, Ph.D.	2006	National Institute of Occupational Safety and Health

Paul Grivas, MPH, Ph.D., JD	2007	Law Practice
Jeff Crane, Ph.D.	2007	University of Miami, Florida
Giffe Johnson, Ph.D.	2007	University of South Florida, Tampa
Malek Khlifi, MD, Ph.D.	2008	New York Medical Center
Robin DeHate, MPH, Ph.D.	2009	GEI Consultants, Tampa, FL

GRADUATE TRAINING - POSTDOCTORAL

	Years of Study	Present Address
Michael Evans, Ph.D.	1973-1976	Amer. Inst. Toxicology Indianapolis, Indiana
Chandrahar Dwivedi, Ph.D.	1973-1976	Meharry Medical College Dept. of Pediatrics Nashville, Tennessee
Richard P. Koshakji, Ph.D.	1973-1976	Dept. of Pharmacology Vanderbilt Medical Center Nashville, Tennessee
James S. MacDonald	1975-1977	Merck Institute for Therapeutic Research West Point, Pennsylvania
Daniel Goodman, Ph.D.	1978-1980	Univ. of Arkansas Med. Sci. Div. of Interdis. Toxicology Little Rock, Arkansas
Robert C. James, Ph.D.	1979-1981	TERRA, Inc. Tallahassee, FL
Peter Wells, Pharm.D.	1979-1981	University of Toronto College of Pharmacy Toronto, Ontario, Canada
Shahata El-Sewedy, Ph.D.	1982-1983	University of Alexandria Medical Research Institute Alexandria, Egypt
Syed F. Ali, Ph.D.	1982-1983	Natl. Ctr. for Toxicol. Res. Jefferson, Arkansas
Jay Gandy, Ph.D.	1986-1987	Univ. of Arkansas Med. Sci.

		Dept. of Pharm. & Tox. Little Rock, Arkansas
Christopher Borgert, Ph.D.	1991-1992	Univ. of Florida Dept. of Pathology Gainesville, Florida
Robert Demott, Ph.D.	1993-1994	Environ Tampa, Florida
Todd Stedeford, Ph.D.	1999-2002	University of Florida School of Law
M. Rony Francois, M.D., Ph.D.	2000-2003	Secretary of Health State of Florida

PUBLICATIONS

1. Harbison, R.D., R.C. Boerth and J.L. Spratt. Quantitative determination of free and conjugated bilirubin by diazo coupling and a liquid-extraction and column-chromatographic technique. Biochem. J. 104:46c-47c, 1967.

2. Harbison. R.D. and J.L. Spratt. Novobiocin-induced hyperbilirubinemia and its reduction by phenobarbital pretreatment. Toxicol. Appl. Pharmacol. 11:257-263, 1967.

3. Harbison, R.D. and J.L. Spratt. Disappearance of plasma bilirubin fractions in the rat after phenobarbital. Arch. Int. Pharmacodyn. 172(1):32-36, 1968.

4. Harbison, R.D. and B.A. Becker. Barbiturate mortality in hypothyroid and hyperthyroid rats. J. Pharm. Sci. 58(2):183-185, 1969.

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6. Harbison, R.D. and B.A. Becker. Relation of dosage and time of administration of diphenylhydantoin and its teratogenic effect in mice. Teratology 2:305-311, 1969.

7. Eling, T.E., R.D. Harbison, B.A. Becker and J.R. Fouts. Diphenylhydantoin effect on neonatal and adult rat hepatic drug metabolism. J. Pharmacol. Exp. Ther. 171:127-137, 1970.

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9. Harbison, R.D. and B.A. Becker. Effect of phenobarbital and SKF 525-A pretreatment on diphenylhydantoin teratogenicity in mice. J. Pharmacol. Exp. Ther. 171:283-288, 1970.

10. Harbison, R.D. and B.A. Becker. Effect of phenobarbital or SKF 525-A pretreatment on diphenylhydantoin deposition in pregnant mice. Toxicol. Appl. Pharmcol. 20:573-581, 1971.

11. Harbison, R.D. and B. Mantilla-Plata. Prenatal toxicity, maternal distribution and placental transfer of tetrahydrocannabinol. J. Pharmacol. Exp. Ther. 180(2):446-453, 1972.

12. Harbison, R.D. and B.A. Becker. Diphenylhydantoin teratogenicity in rats. Toxicol. Appl. Pharmacol. 22:193-200, 1972.

13. Wilson, B.J. and R.D. Harbison. Rubratoxins. J. Am. Vet. Med. Assoc. 163(11): 1274-1276, 1973.

14. Koshakji, R.P., B.J. Wilson and R.D. Harbison. Effect of rubratoxin B on prenatal growth and development in mice. Res. Comm. Chem. Pathol. Pharmacol. 5:584-593, 1973.

15. Koshakji, R.P., J. Cole and R.D. Harbison. Influence of injection volume on parathion toxicity and plasma and brain cholinesterase inhibition. Res. Comm. Chem. Pathol. Pharmacol. 6(2):677-687, 1973.

16. Mantilla-Plata, B. and R.D. Harbison. Effect of phenobarbital and SKF 525-A pretreatment, sex, liver injury and vehicle on Δ^9 tetrahydrocannabinol toxicity. Toxicol. Appl. Pharmacol. 27:123-130, 1974.

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32. Mantilla-Plata, B., G.L. Clewe and R.D. Harbison. Δ^9-tetrahydrocannabinol-induced changes in prenatal growth and development of mice. Toxicol. Appl. Pharmacol. 33:333-340, 1975.

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34. Mantilla-Plata, B. and R.D. Harbison. Influence of alteration of THC metabolism on THC-induced teratogenesis. In: The Pharmacology of Marijuana, Raven Press, pp. 733-742, 1975.

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56. Evans, M.A. and R.D. Harbison. Cocaine-induced hepatotoxicity in mice. Toxicol. Appl. Pharmacol. 45:739-754, 1978.

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65. Olson, R.D., J.S. MacDonald, C.V. vanBoxtel, R.C. Boerth, R.D. Harbison, A.E. Slonim, R.W. Freeman and J.A. Oates. Regulatory role of glutathione and soluble sulfhydryl groups in the toxicity of adriamycin. J. Pharmacol. Exp. Ther. 215(2):450-454, 1980.

66. Harbison, R.D., W.W. Eckenfelder, G. Gallagher and G. Moein. National Hazardous Materials Training Program. Proceedings of 1980 National Conference on Control of Hazardous Material Spills, 492-494, 1980.

67. Clarke, J.H., F.G. Ziegler, R.D. Harbison and R.C. James. A Model for Assessment of Environmental Impact of Hazardous Materials Spills and Leaching. Proceedings of 1980 National Conference on Control of Hazardous Material Spills, pp. 293-295, 1980.

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ABSTRACTS
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